Alerts, Notices, and Case Reports

Acute Onset of Painful Ophthalmoplegia Following Chiropractic Manipulation of the Neck
Initial Sign of Intracranial Aneurysm

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NEUROLOGIC COMPLICATIONS following chiropractic manipulation of the neck, although uncommon, have been well documented in the literature.[1-10] The most frequently reported adverse effect is posterior circulation stroke associated with vertebral artery dissection. Less frequently reported complications include anterior circulation stroke due to extracranial internal carotid dissection, aneurysm, or hematoma and cervical disc herniation resulting in spinal cord compression and radiculopathies. Cases of meningeal hematoma and diaphragmatic paralysis following neck manipulation also have been identified.

The case presented here illustrates a temporal association between cervical manipulation and the clinical declaration of a previously asymptomatic posterior communicating artery aneurysm. This is the first known report of such a complication related to chiropractic manipulation of the cervical spine.

Report of a Case

The patient, a 45-year-old female waiter, visited her chiropractor for symptoms of left neck stiffness and occipital head pain. She previously had undergone cervical manipulations for the treatment of tension headache and neck muscle spasms. On this occasion, she attributed the occipital and neck pain to muscle spasm. Following the manipulation of her neck, which reportedly included rapid rotation to the right and hyperextension, she experienced a sudden onset of searing retro-orbital pain on the left. Over the next 36 hours, complete left eye ptosis developed, and she was unable to adduct, elevate, or depress her left eye. The pain in her neck and occiput worsened, which in addition to her new focal neurologic symptoms, led to the initial medical evaluation four days after the neck manipulation. The patient was found to be normotensive with unremarkable general physical findings. The neurologic examination was notable for a pupil-involving oculomotor paresis on the left and a subtle left lateral rectus muscle paresis. A computed tomographic (CT) scan of the head with contrast proved to be unremarkable. She was diagnosed with presumptive herpes zoster, and a regimen of acyclovir (Zovirax) was started. She then was referred to our institution for further evaluation of oculomotor nerve paresis.

Her visual acuity was 20/20 in the right eye, 20/30 in the left eye, and visual fields were full to confrontation. A funduscopic examination revealed sharp discs with no retinal lesions. The left pupil was dilated and sluggishly responsive to light, accompanied by a complete left ptosis. The patient could not elevate, depress, or adduct the left eye, but was able to intort the eye on downward gaze. Abduction of the left eye was moderately compromised, possibly related to a remote history of the surgical correction of strabismus. Facial sensation was completely preserved, and the remainder of her cranial nerves on sequential testing were normal. The right eye and the results of her ophthalmologic and neurologic examinations were otherwise normal. No cervical or cranial bruits were detected.

A second head CT scan without contrast on admission was unremarkable. This study was followed by a brain magnetic resonance imaging study with and without gadolinium contrast. This study also was unremarkable, with normal vascular flow. Cerebrospinal fluid analysis on admission showed no substantial evidence of blood and no xanthochromia. The initial findings of abducens nerve paresis in addition to the oculomotor palsy raised the suspicion of a dissection of the left internal carotid artery following neck manipulation, with possible extension into its intracavernous segments. An angiogram of the intracranial vessels using three-dimensional (3D) multiple overlapping thin-slab acquisition (MOTSA) and time-of-flight (TOF) high-resolution magnetic resonance (MR) and a two-dimensional TOF MR angiogram of the cervical vessels were obtained (not shown). No evidence of dissection, stenosis, or aneurysm was found in either intracranial carotid artery systems or their major branches or in the vertebrobasilar system. The brain MR imaging study was repeated with fine-cut imaging through the orbital apices, and the cavernous sinus showed a thickening of the intracavernous segment of the left internal carotid artery (Figure 1). The question of dissection within the intracavernous segment of the internal carotid artery was addressed further with a 3D MOTSA high-resolution MR angiogram (Figure 2). This study identified a saccular aneurysm at the origin of the left posterior communicating artery with extension inferiorly from its takeoff point. A sagittal 3D TOF MR angiogram confirmed the presence of a solitary bilobed aneurysm with approximate dimensions of 5 × 7 mm (Figure 3). A cerebral angiogram further showed this aneurysm as measuring about 8 mm in its longest dimension and identified no additional intracranial aneurysms (Figure 4).

A left pterional craniotomy was performed, and the

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ABBREVIATIONS USED IN TEXT

3D = three-dimensional
CT = computed tomographic
MOTSA = multiple overlapping thin-slab acquisition [MR angiogram]
MR = magnetic resonance
TOF = time-of-flight

posterior communicating artery aneurysm was exposed. The aneurysm was found to have an extremely thin-walled region through which turbulent blood flow was observed. No evidence of subarachnoid hemorrhage was identified, and the aneurysm was clipped successfully. The patient had an uneventful postoperative course with resolution of her head pain, but with continued residual oculomotor palsy a month following the aneurysmal clipping.

Discussion

Saccular aneurysms typically present in adults between the ages of 40 and 60 years, most commonly at bifurcation points of arteries off the circle of Willis. The most frequent locations include the anterior communicating artery (30% to 35%), the internal carotid artery at the takeoff of the posterior communicating artery (30% to 35%), and the middle cerebral arteries (20%).[11,12] These sites represent 90% of all intracerebral aneurysms, with posterior circulation aneurysms making up the remaining 10%.[13]

Unruptured aneurysms, particularly of the internal carotid artery, may cause signs and symptoms similar to a mass lesion, stroke, or a new onset of seizures.[14] Although thunderclap headache is a classical feature of rupture, it may also be a feature of acute aneurysmal expansion or thrombosis.[15-17] These findings in the absence of hemorrhage have been described, with the onset of headache ascribed to probable local thrombosis or acute morphologic changes in the vessel wall.[18]

The natural history of untreated aneurysms is characterized by progressive enlargement. Those lesions located

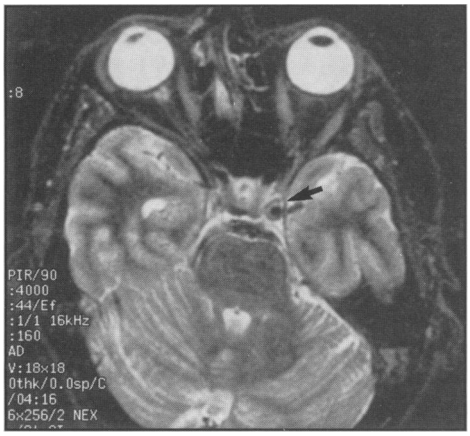

Figure 1.—An axial T2-weighted magnetic resonance scan shows a hypointense center with signal heterogeneity suggestive of turbulent flow in the left posterior communicating artery aneurysm **(arrow)**.

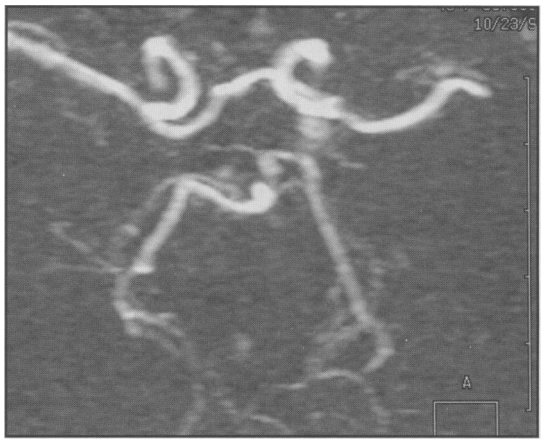

Figure 2.—An axial multiple overlapping thin-slab acquisition magnetic resonance angiogram shows the aneurysm of the left posterior communicating artery.

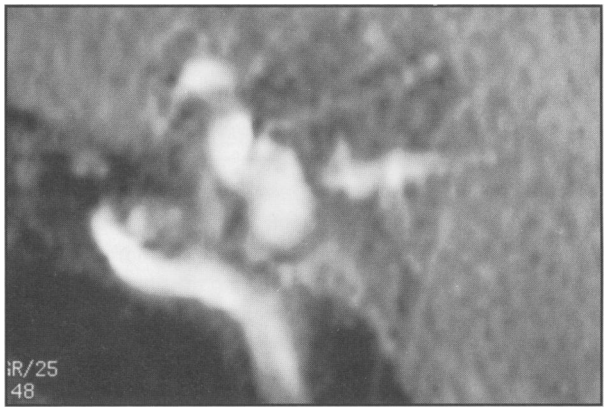

Figure 3.—A 3-dimensional time-of-flight magnetic resonance angiogram shows high signal within the inferiorly directed aneurysm located at the takeoff of the left posterior communicating aneurysm.

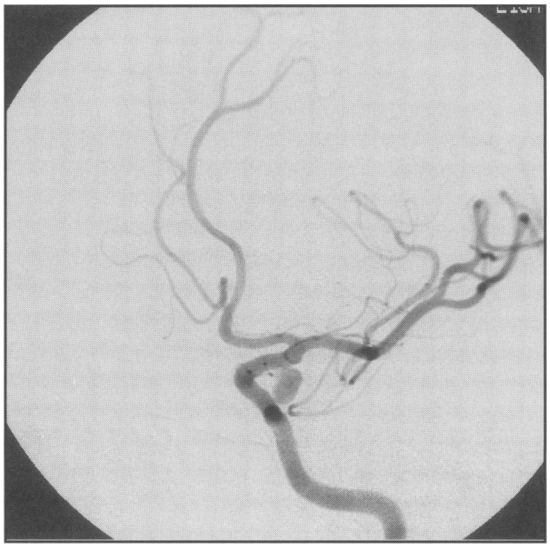

Figure 4.—A left internal carotid artery cerebral angiogram shows a bilobed saccular aneurysm arising from the left posterior communicating aneurysm.

at bifurcation points with the internal carotid artery have particularly turbulent intravascular flow dynamics and sheer forces within the aneurysmal sac.[19-21] Although most authors accept that the size of an aneurysm is the most important factor predicting rupture, no level has been firmly established below which subarachnoid hemorrhage has not occurred.[22] Several studies report that the annual risk of rupture of an intact aneurysm remains at least 1%.[23] Isolated cranial nerve palsies, and specifically oculomotor nerve palsy, have been described in association with progressive aneurysmal enlargement of the posterior communicating artery and with sudden dilation of an existing aneurysmal sac in the absence of subarachnoid bleeding.[24] Although a pupil-involving oculomotor paresis is the typical presentation of a posterior communicating artery aneurysm, pupil-sparing partial oculomotor paresis may represent an ischemic insult to the nerve or, uncommonly, an initial presentation of an intracranial aneurysm. In these cases, iridoplegia generally becomes apparent within days to weeks following the acute insult.[25-27] Okawara reported that the average time to rupture of a posterior communicating artery aneurysm presenting with oculomotor paresis was 29.6 days (based on 6 case reports).[18] Clearly, the evolution of iridoplegia in a patient with ophthalmoplegia should prompt early noninvasive neuroimaging studies and cerebral angiogram, especially in patients younger than 50 years.

Aneurysm formation on intracranial segments of the internal carotid artery has been reported following skull-based fractures and traumatic hyperextension and rotation of the neck.[28] Blunt impact to the frontal or temporal aspects of the skull also has been associated with acquired intracranial aneurysm, particularly of the pericallosal artery. The mechanism here is thought to be the development of shearing forces between the inferior free margin of the falx cerebri and the distal anterior cerebral artery.[29] Two case studies were reported in which a clinically silent posterior communicating artery aneurysm located at the bifurcation with the internal carotid artery declared itself acutely as a painful oculomotor palsy after minor head trauma.[30] In those two cases, the aneurysm acted as a mass lesion stretching and compressing the adjacent oculomotor nerve, resulting in acute ophthalmoplegia.

In the case presented here, chiropractic manipulation of the neck, including rotation and hyperextension, preceded the acute onset of severe periorbital and retro-orbital pain. These symptoms evolved to complete ipsilateral oculomotor palsy involving the pupil. The clinical history identified no evidence of preceding head or neck trauma except for the associated chiropractic manipulation. Both upper and lower cranial nerve palsies have been described recently in patients with acute extracranial dissection of the internal carotid artery.[31] Thus, given the presenting symptoms of hemicrania and acute oculomotor and possibly new abducens pareses, the initial radiologic studies were directed toward identifying an internal carotid artery dissection, with special attention paid to the artery at the skull base. When the radiologic studies were reviewed, neither a dissection nor an aneurysm was identified. Identification of the posterior communicating artery aneurysm was possible only after including additional fine-cut views through the cavernous sinus. The aneurysm was visible as a flow void on T1- and T2-weighted MR images and subsequently identified on MR angiogram of the intracavernous portion of the internal carotid artery.

In the case reported here, transient changes of blood flow through the posterior communicating artery aneurysm induced by neck manipulation may have contributed to its acute expansion inferiorly and thus to the rapid onset of oculomotor palsy. This case underscores the need to include a conclusive evaluation for a previously asymptomatic intracranial aneurysm as an underlying cause of an acutely acquired painful pupil-involving oculomotor palsy in a patient with minor neck trauma.

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